(FILE 'HCAPLUS' ENTERED AT 11:58:48 ON 21 FEB 2003)
L1 17728 SEA FILE=HCAPLUS ABB=ON PLU=ON ADD(10A)(ATTENTION DEFICIT) OR ADHD OR ATTENTION(3W)DISORDER OR AUTISM OR PARKINSON? OR PDD OR PERVAS? DEVELOP? DISORDER OR DYSAUTONOM? OR DYS AUTONOM? OR SIDS OR SUDDEN INFANT DEATH SYNDROME OR AUTISTIC
L2 9 SEA FILE=HCAPLUS ABB=ON PLU=ON L1 AND PYLORI

L2 ANSWER 1 OF 9 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:905855 HCAPLUS
DOCUMENT NUMBER: 138:303
TITLE: Caspase inhibitors and therapeutic uses
INVENTOR(S): Mortimore, Michael; Miller, Andrew; Studley, John; Charrier, Jean-Damien
PATENT ASSIGNEE(S): Vertex Pharmaceuticals Incorporated, USA
SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002094263	A2	20021128	WO 2002-US16353	20020523

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-292969P P 20010523
OTHER SOURCE(S): MARPAT 138:303
AB This invention provides compds. which are effective inhibitors of apoptosis and IL-1.beta. secretion. The invention also discusses the therapeutic potential of these compds. in treating diseases like IL-1 mediated disease, apoptosis mediated disease or an inflammatory disease.

L2 ANSWER 2 OF 9 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:857965 HCAPLUS
TITLE: Helicobacter **pylori** - does it only cause gastroduodenal disease?
AUTHOR(S): Wlodarek, Dariusz; Pakszys, Waldemar; Barlik, Magdalena
CORPORATE SOURCE: Zakl. Dietetyki, Katedra Dietetyki i Zywnosci Funkcjonalnej, Wydz. Nauk o Zywieniu Czlowieka i Konsumpcji, SGGW, Warsaw, Pol.
SOURCE: Polski Merkuriusz Lekarski (2001), 11(65), 456-459
CODEN: PMLOB9; ISSN: 1426-9686
PUBLISHER: Medpress
DOCUMENT TYPE: Journal

LANGUAGE: Polish

AB Helicobacter **pylori** is a human pathogen that can be found all over the world. It is responsible for the following diseases of gastrointestinal tube: gastritis, gastric ulcer, duodenal ulcer, gastric cancer, gastric lymphomas, Menetier disease. Some research has been done recently trying to identify the connection between H. **pylori** infection and idiopathic **Parkinson**'s Disease morbidity. Some of them show that people with this neurol. disease are more likely to have ulcers and also seropositivity in the direction of H. pylon. The direct influence of H. **pylori** infection on **Parkinson** Disease is not known but the following relations are suggested: H. pylon may produce toxins that damage substantia negra in brain; possible cross reaction of h. **pylori** antibodies with dopaminergic neurons; indirect influence of antacids contg. aluminum used to alleviate the symptoms of ulcers. Investigations of the reasons for idiopathic **parkinson** disease draw attention to the influence of food factors. some researches show that there is a relation between the frequency of eating certain foods and the **parkinson** disease morbidity we have numerous techniques that allow us to diagnose h. **pylori** infection. those techniques have different sensitivity, accuracy, invasiveness and costs, which dets. their usefulness in clin. diagnostics. Approach to eradication of bacteria is still discussed because H. **pylori** infection doesn't always lead to health problems. Polish Working Group on Helicobacter **pylori**, called by the National Consultant's Team on Gastroenterol. explained clearly when eradication is advisable and when it can be waived.

L2 ANSWER 3 OF 9 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:555358 HCAPLUS
DOCUMENT NUMBER: 137:114486
TITLE: Novel receptors for Helicobacter **pylori** and use thereof
INVENTOR(S): Miller-Podraza, Halina; Teneberg, Susann; Angstroem, Jonas; Karlsson, Karl-Anders; Natunen, Jari
PATENT ASSIGNEE(S): Carbion Oy, Finland
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002056893	A1	20020725	WO 2002-FI43	20020118

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,

SN, TD, TG

FI 2001000118	A	20020720	FI 2001-118	20010119
WO 2003002128	A1	20030109	WO 2002-FI575	20020628

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

FI 2001-118	A	20010119
FI 2001-1403	A	20010629
WO 2002-FI43	A	20020118

AB The present invention describes a substance or a receptor comprising Helicobacter **pylori**-binding oligosaccharide sequence [Gal(A)q(NAc)r/Glc(A)q(NAc)r.alpha.3/.beta.3]s[Gal.beta.4GlcNAc.beta.3]tGal.beta.4Glc(NAc)u wherein q, r, s, t, and u are each independently 0 or 1, and the use thereof in, e.g., pharmaceutical and nutritional compns. for the treatment of conditions due to the presence of Helicobacter **pylori**. The invention is also directed to the use of the receptor for diagnostics of Helicobacter **pylori**.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 9 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:488136 HCAPLUS
DOCUMENT NUMBER: 137:30245
TITLE: Methods for diagnosing **pervasive development disorders, dysautonomia** and other neurological conditions
INVENTOR(S): Fallon, Joan M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 9 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002081628	A1	20020627	US 2001-990909	20011116

PRIORITY APPLN. INFO.: US 2000-249239P P 20001116

AB Methods for aiding in the diagnosis of disorders including, but not limited to, **PDDs** (**Pervasive Development Disorders**), **Dysautonomic** disorders, **Parkinson**'s disease and **SIDS** (**Sudden Infant Death Syndrome**). In one aspect, a diagnosis method comprises analyzing a stool sample of an individual for the presence of a biol. marker (or marker compd.) comprising one or more pathogens, which provides an indication of whether the individual has, or can develop, a disorder including, but not limited to, a **PDD, Dysautonomia,**

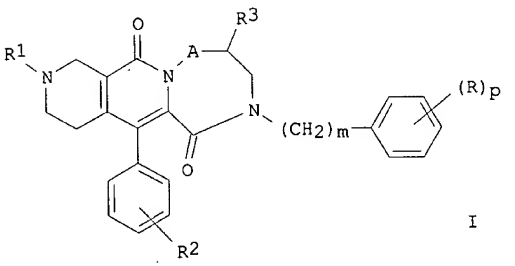
Parkinsons disease and **SIDS**. Preferably, the presence of one or more pathogens is detd. using a stool immunoassay to det. the presence of antigens in a stool sample, wherein such antigens are assocd. with one or more pathogens including, but not limited to, Giardia, Cryptosporidium, E. histolytica, C. difficile, Adenovirus, Rotavirus or H. **pylori**.

L2 ANSWER 5 OF 9 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:220552 HCAPLUS
DOCUMENT NUMBER: 136:247613
TITLE: Preparation of tricyclic heterocyclic compounds as tachykinin receptor antagonists
INVENTOR(S): Ikeura, Yoshinori; Hashimoto, Tadatoshi; Tarui, Naoki; Kamo, Izumi; Shirai, Junya
PATENT ASSIGNEE(S): Takeda Chemical Industries, Ltd., Japan
SOURCE: PCT Int. Appl., 84 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002022574	A1	20020321	WO 2001-JP7815	20010910
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001086188	A5	20020326	AU 2001-86188	20010910
JP 2002155084	A2	20020528	JP 2001-274336	20010910

PRIORITY APPLN. INFO.: JP 2000-280154 A 20000911
WO 2001-JP7815 W 20010910

OTHER SOURCE(S): MARPAT 136:247613
GI



I

AB The title compds. I [A = (CH2)n ; R represents hydrogen, halo, etc.; R1 represents hydrogen, optionally substituted alkyl, aryl, acyl, alkoxycarbonyl, carbamoyl, mono- or dialkylcarbamoyl, or alkylsulfonyl; R2 represents hydrogen, halogeno, or optionally halogenated alkyl; R3 represents hydrogen or alkyl; R represents hydrogen, halogeno, optionally halogenated alkyl, or optionally halogenated alkoxy; m is an integer of 0 to 3; n is 1 or 2; and p is an integer of 0 to 3; a proviso is given] are prepd. I are useful in the treatment of urination disorder. Processes for prepg. I are claimed. In an in vitro test for substance P antagonism, compds. of this invention showed IC50 of 0.0164 nM to 0.0762 nM. Formulations are given.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:911965 HCAPLUS
DOCUMENT NUMBER: 136:199369
TITLE: **Sudden infant death syndrome** and enteric infection
AUTHOR(S): Reid, G. M.
CORPORATE SOURCE: Te Aroha, N. Z.
SOURCE: Medical Hypotheses (2001), 57(5), 580-582
CODEN: MEHYDY; ISSN: 0306-9877
PUBLISHER: Churchill Livingstone
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. The assocn. of Helicobacter **pylori** in the stomach, trachea and lungs with the incidence of **SIDS**, gastric ulcers and cancer may have a counterpart in animals. In field studies of white muscle disease (WMD) and hepatic necrosis in selenium-deficient pigs dying suddenly, veterinarians identified gastric ulcers in 40% of inspected piglets. The lesion was also commonly obsd. by researchers in exptl. produced vitamin E-selenium deficiency and other researchers suspected that gastric ulcers in swine may be assocd. with vitamin E-selenium deficiency. Mice preferentially concd. 75selenium in peritoneal exudative cells (PEC) when 75selenium as selenium selenate was administered by stomach tube to selenium-deficient mice. Selenium concd. in PECs as glutathione peroxidase (GSHPx). GSHPx-deficient leukocytes in peritoneal exudate failed to kill yeast cells. GSHPx deficiency has also been assocd. with decreased microbicidal activity of leukocytes in patients with chronic granulomatosis. The selenium-deficient swine were usually growing rapidly in crowded conditions, and, apart from WMD and hepatic necrosis, edema was prominent in the spiral colon, s.c. tissues, lungs and submucosa of the stomach. The elevated immunol. response in the spleen and lungs of **SIDS** victims suggests an initial defective microbicidal propensity of the peritoneal exudative cells.

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 7 OF 9 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:635933 HCAPLUS
DOCUMENT NUMBER: 135:215973

TITLE: Use of peptide conjugates for enhancing drug delivery across biological membranes and tissues
INVENTOR(S): Rothbard, Jonathan B.; Wender, Paul A.
PATENT ASSIGNEE(S): Cellgate, Inc., USA
SOURCE: PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001062297	A1	20010830	WO 2001-US4459	20010209
WO 2001062297	C2	20030109		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002009491	A1	20020124	US 2001-779693	20010207
EP 1263469	A1	20021211	EP 2001-909135	20010209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.:
US 2000-182166P P 20000214
US 2001-779693 A 20010207
WO 2001-US4459 W 20010209

AB This invention provides compns. and methods for enhancing delivery of drugs and other agents across a biol. barrier, including epithelial tissues such as the skin, gastrointestinal tract, pulmonary epithelium, and the like. The compns. and methods are also useful for delivery across endothelial tissues, including the blood brain barrier. The compns. and methods employ a delivery enhancing transporter that has sufficient guanidino or amidino sidechain moieties to enhance delivery of a compd. across one or more layers of the tissue, compared to the non-conjugated compd. The delivery-enhancing polymers include, for example, poly-arginine mols. that are preferably between about 6 and 50 residues in length. Taxol conjugates with a heptamer of arginine was sol. in water in contrast with taxol itself. The conjugate was equally potent when assayed for biol. activity using std. cytotoxicity assay.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 8 OF 9 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:452866 HCAPLUS
DOCUMENT NUMBER: 135:71250
TITLE: Novel Helicobacter **pylori**-binding substances and use thereof
INVENTOR(S): Karlsson, Karl-anders; Leonardsson, Irene; Teneberg, Susann; Angstroem, Jonas

PATENT ASSIGNEE(S): A+ Science Invest AB, Swed.
SOURCE: PCT Int. Appl., 88 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001043751	A1	20010621	WO 2000-SE2567	20001215
W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1237558	A1	20020911	EP 2000-987920	20001215
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
NO 2002002890	A	20020815	NO 2002-2890	20020617

PRIORITY APPLN. INFO.: SE 1999-4581 A 19991215
WO 2000-SE2567 W 20001215

OTHER SOURCE(S): MARPAT 135:71250

AB Helicobacter **pylori**-binding substances comprising Gal.beta.3GlcNAc or Gal.beta.3GalNAc are described, as well as use thereof in pharmaceutical compns. and food-stuff, and methods for treatment of conditions due to the presence of Helicobacter **pylori**. Also use of said substance for the identification of bacterial adhesions, for the prodn. of a vaccine against Helicobacter **pylori**, for diagnosis of Helicobacter **pylori** infections, for typing of Helicobacter **pylori** , for identification of Helicobacter **pylori** binding substances and for inhibition of the binding of Helicobacter **pylori** is described.

REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 9 OF 9 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:79969 HCAPLUS
DOCUMENT NUMBER: 135:44211
TITLE: Review: mitochondrial medicine - molecular pathology of defective oxidative phosphorylation
AUTHOR(S): Fosslien, Egil
CORPORATE SOURCE: Department of Pathology, University of Illinois, Chicago, IL, 60612, USA
SOURCE: Annals of Clinical and Laboratory Science (2001), 31(1), 25-67
CODEN: ACLSCP; ISSN: 0091-7370
PUBLISHER: Association of Clinical Scientists
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 322 refs. Different tissues display distinct sensitivities to defective mitochondrial oxidative phosphorylation (OXPHOS). Tissues highly dependent on O such as the cardiac muscle, skeletal and smooth muscle, the central and peripheral nervous system, the kidney, and the insulin-producing pancreatic .beta.-cell are esp. susceptible to defective OXPHOS. There is evidence that defective OXPHOS plays an important role in atherogenesis, in the pathogenesis of Alzheimer's disease, **Parkinson**'s disease, diabetes, and aging. Defective OXPHOS may be caused by abnormal mitochondrial biosynthesis due to inherited or acquired mutations in the nuclear (n) or mitochondrial (mt) DNA. For instance, the presence of a mutation of the mtDNA in the pancreatic .beta.-cell impairs ATP (ATP) generation and insulin synthesis. The nuclear genome controls mitochondrial biosynthesis, but mtDNA has a much higher mutation rate than nDNA because it lacks histones and is exposed to the radical O species (ROS) generated by the electron transport chain, and the mtDNA repair system is limited. Defective OXPHOS may be caused by insufficient fuel supply, by defective electron transport chain enzymes (Complexes I-IV), lack of the electron carrier coenzyme Q10, lack of oxygen due to ischemia or anemia, or excessive membrane leakage, resulting in insufficient mitochondrial inner membrane potential for ATP synthesis by the F0F1-ATPase. Human tissues can counteract OXPHOS defects by stimulating mitochondrial biosynthesis; however, above a certain threshold the lack of ATP causes cell death. Many agents affect OXPHOS. Several nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit or uncouple OXPHOS and induce the 'topical' phase of gastrointestinal ulcer formation. Uncoupled mitochondria reduce cell viability. The Helicobacter **pylori** induces uncoupling. The uncoupling that opens the membrane pores can activate apoptosis. Cholic acid in exptl. atherogenic diets inhibits Complex IV, cocaine inhibits Complex I, the poliovirus inhibits Complex II, ceramide inhibits Complex III, azide, cyanide, chloroform, and methamphetamine inhibit Complex IV. EtOH abuse and antiviral nucleoside analog therapy inhibit mtDNA replication. By contrast, melatonin stimulates Complexes I and IV and Gingko biloba stimulates Complexes I and III. Oral Q10 supplementation is effective in treating cardiomyopathies and in restoring plasma levels reduced by the statin type of cholesterol-lowering drugs.

REFERENCE COUNT: 322 THERE ARE 322 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 12:01:37 ON 21 FEB 2003)

L1 17728 SEA FILE=HCAPLUS ABB=ON PLU=ON ADD(10A)(ATTENTION DEFICIT) OR ADHD OR ATTENTION(3W)DISORDER OR AUTISM OR PARKINSON? OR PDD OR PERVAS? DEVELOP? DISORDER OR DYSAUTONOM? OR DYS AUTONOM? OR SIDS OR SUDDEN INFANT DEATH SYNDROME OR AUTISTIC

L9 39913 SEA L1(S)(DETERM? OR DETECT? OR DET## OR SCREEN? OR DIAGNOS?)

L10 30 SEA L9 AND PYLORI

PROCESSING COMPLETED FOR L10

L11 15 DUP REM L10 (15 DUPLICATES REMOVED)

L11 ANSWER 1 OF 15 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-713307 [77] WPIDS
DOC. NO. CPI: C2002-202163
TITLE: New receptor useful e.g. in the treatment of gastric ulcer comprises Helicobacter **pylori** binding oligosaccharide sequence.
DERWENT CLASS: B04
INVENTOR(S): ANGSTROEM, J; KARLSSON, K; MILLER-PODRAZA, H; TENEBERG, S; NATUNEN, J
PATENT ASSIGNEE(S): (CARB-N) CARBION OY
COUNTRY COUNT: 100
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002056893	A1	20020725	(200277)*	EN	75

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW

FI 2001000118 A 20020720 (200277)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002056893	A1	WO 2002-FI43	20020118
FI 2001000118	A	FI 2001-118	20010119

PRIORITY APPLN. INFO: FI 2001-118 20010119
AN 2002-713307 [77] WPIDS
AB WO 200256893 A UPAB: 20021129
NOVELTY - A substance comprising Helicobacter **pylori** binding oligosaccharide sequence (I) is used in the production of a composition having H. **pylori** binding or inhibiting activity.

DETAILED DESCRIPTION - Production of a composition having binding activity to Helicobacter **pylori** involves the use of a substance comprising H. **pylori** binding oligosaccharide sequence of formula (I) and its analogs or derivatives.

(Gal(A)q(NAc)r/Glc(A)q(NAc)r alpha 3/ beta 3)s(Gal beta 4GlcNAc beta 3)tGal beta 4Glc(NAc)u (I).

q - u = 0 or 1;

when t is 0 and u is 0, the oligosaccharide sequence is linked to a polyvalent carrier or present as a free oligosaccharide in high concentration.

INDEPENDENT CLAIMS are also included for:

(1) A H. **pylori** binding substance comprising an oligosaccharide sequence Glc(A)q(NAc)r alpha 3/ beta 3Gal beta 4Glc(NAc)u; or GalA(NAc)r alpha 3/ beta 3Gal beta 4Glc(NAc)u or their analogs or derivatives, provided that when the oligosaccharide sequence contains beta 3 linkage, both q and r are 0 or 1 and r and u are 0 or 1; and

(2) A H. **pylori** binding substance comprising the oligosaccharide sequence (Gal(A)q(NAc)r/Glc(A)q(NAc)r alpha 3/ beta 3)Gal beta 4Glc(NAc)u (II) and its analogs or derivatives, provided that (II) is not Gal alpha 3Gal beta 4Glc/GlcNAc.

q, r and u = 0 or 1.

ACTIVITY - Antibacterial; Antiviral; Antiinflammatory; Antiulcer; Cytotoxic; Hepatotropic; Dermatological; Cardiant; Immunosuppressive; Antianemic.

MECHANISM OF ACTION - H. **pylori** binder or inhibitor.

USE - For the production of a pharmaceutical composition (preferably medicament) for the treatment or prophylaxis of any condition due to the presence of Helicobacter **pylori** such as in the gastrointestinal tract of a patient, chronic superficial gastritis, gastric ulcer, duodenal ulcer, gastric adenocarcinoma, non-Hodgkin lymphoma in human stomach, liver disease, pancreatic disease, skin disease, heart disease, or autoimmune diseases including autoimmune gastritis and pernicious anaemia and non-steroid anti-inflammatory drug (NSAID) related gastric disease, or for prevention of **sudden infant death syndrome**; for the **diagnosis** of a condition due to infection by H. **pylori**; for production of a nutritional additive or composition for the treatment or prophylaxis of any condition due to the presence of H. **pylori** (preferably in an infant food); for the identification of bacterial adhesion; for the production of a vaccine against H. **pylori**; for binding bacterial viruses and toxin (preferably a toxin of Clostridium difficile (all claimed).

ADVANTAGE - The substance has a significant binding specificity to H. **pylori**, compared to the prior art sequences. The binding of the bacterium to the sequence was very reproducible, though the general saccharide bindings of H. **pylori** suffer from phase variations of the bacterium. The high affinity of the binding was visible in the overlay assay even at low picomolar amounts of the glycolipid sequences.

Dwg.0/11

L11 ANSWER 2 OF 15 WPIDS (C) 2003 THOMSON DERWENT

ACCESSION NUMBER: 2002-500127 [53] WPIDS

DOC. NO. CPI: C2002-141599

TITLE: Determining susceptibility of a human to a disease associated with A2a receptor functional hyperactivity or reduced A2a receptor activity comprises detecting the presence of a polymorphism in the A2a receptor or A2a receptor gene.

DERWENT CLASS: B04 D16

INVENTOR(S): DOWELL, S J; SHEEHAN, M J

PATENT ASSIGNEE(S): (GLAX) GLAXO GROUP LTD

COUNTRY COUNT: 97

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002036816	A2	20020510	(200253)*	EN	44

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ

NO NZ PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2002010761 A 20020515 (200258)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002036816 A2		WO 2001-GB4865	20011102
AU 2002010761 A		AU 2002-10761	20011102

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002010761 A	Based on	WO 200236816

PRIORITY APPLN. INFO: GB 2000-29577 20001202; GB 2000-26945 20001103

AN 2002-500127 [53] WPIDS

AB WO 200236816 A UPAB: 20020820

NOVELTY - Determining susceptibility of a human subject to a disease associated with A2a receptor functional hyperactivity or reduced A2a receptor activity comprises detecting if a polymorphism of the A2a receptor gene exists in a nucleic acid sample or if a polymorphism of the A2a receptor exists in a protein sample. The presence of the polymorphism indicates susceptibility.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) methods of treating an A2a receptor associated disease in a human subject comprises detecting if a polymorphism of the A2a receptor gene exists in a nucleic acid sample, or if a polymorphism of the A2a receptor exists in a protein sample from the subject, and administering an A2a agonist, inverse agonist or antagonist to the subject;

(2) patient packs comprising an A2a agonist, inverse agonist or antagonist and instructions for administration of the agonist, inverse agonist or antagonist to a human subject determined to have a polymorphism of the A2a receptor or A2a receptor gene;

(3) a method of treating an A2a receptor associated disease in a human subject having a polymorphism characterized by the presence of guanine at position 1174 on the A2a receptor gene (longer sequence) or at position 1165 on the A2a receptor gene (shorter sequence), by administering an A2a agonist or antagonist; and

(4) a method of treating an A2a receptor associated disease in a human subject having a polymorphism characterized by the presence of glycine at position 392 on the amino acid sequence of the A2a receptor (longer sequence) or at position 389 on the amino acid sequence of the A2a receptor (shorter sequence), by administering an A2a agonist or antagonist.

ACTIVITY - Antiasthmatic; Anti-Parkinsonian; Antiinflammatory; Dermatological; Antibacterial; Antiallergic; Nootropic; Neuroprotective; Cardiovascular; Immunosuppressive; Antiarthritic; Antirheumatic.

No data is given.

MECHANISM OF ACTION - A2a agonist; A2a antagonist.

USE - The A2a agonist, inverse agonist or antagonist is useful in the preparation or manufacture of a medicament for treating an

A2a receptor associated disease in a human subject having a polymorphism of the A2a receptor gene, where the A2a receptor associated disease is a respiratory disorder (e.g. asthma or chronic obstructive pulmonary disorder), a motor dyskinesia disorder (e.g. Parkinson's disease) or a disease associated with deregulation of the immune system. The nucleotide sequence of a human A2a receptor gene polymorphism may be used to identify compounds that affect expression of the human A2a receptor (all claimed). A2a agonists may further be used to treat diseases of the gastrointestinal tract (e.g. inflammatory bowel disease, Helicobacter **pylori** -induced gastritis or intestinal inflammatory diseases secondary to radiation exposure or allergen exposure), psoriasis, allergic dermatitis and hypersensitivity reactions, diseases of the central nervous system which have inflammatory component (e.g. Alzheimer's disease), cardiac conditions (e.g. peripheral vascular disease), or autoimmune disease (e.g. rheumatoid arthritis).
Dwg.0/17

L11 ANSWER 3 OF 15 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-690118 [74] WPIDS
DOC. NO. NON-CPI: N2002-544343
DOC. NO. CPI: C2002-195013
TITLE: **Determining** a disorder or condition e.g., **Parkinson**'s disease, comprises analyzing a stool sample to **determine** presence of a pathogen e.g., Giardia and Cryptosporidium and correlating it with a disorder or lack of disorder.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): FALLON, J M
PATENT ASSIGNEE(S): (FALL-I) FALLON J M
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2002081628	A1	20020627	(200274)*		9

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2002081628	A1 Provisional	US 2000-249239P	20001116
		US 2001-990909	20011116

PRIORITY APPLN. INFO: US 2000-249239P 20001116; US 2001-990909 20011116
AN 2002-690118 [74] WPIDS
AB US2002081628 A UPAB: 20021118
NOVELTY - Determining (M) if an individual has, or can develop, a disorder or condition, comprises obtaining a stool sample from the individual, analyzing the stool sample to determine the presence of a pathogen, and correlating the presence of a pathogen with a disorder or lack of disorder.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a biological marker (I) for determining if an individual has, or can develop, a disorder or condition, comprising a pathogen in a stool sample of the individual.

USE - (M) is useful for **determining** if an individual has, or can develop, a disorder or condition, where the disorder comprises a **pervasive development disorder** (**PDD**), a **dysautonomic** disorder, or a neurological disorder (claimed). (M) is useful for aiding in the **diagnosis** of various human disorders, such as **PDD, dysautonomia, Parkinson's** syndrome, **sudden infant death syndrome** (**SIDS**), etc.

ADVANTAGE - No data existed previously to show a correlation and association between various disorders such as e.g., autism, Parkinson's, ADD (attention deficit disorder), dysautonomia, and the presence of pathogens in an individuals digestive tract.

Dwg.0/4

L11 ANSWER 4 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:586592 BIOSIS
DOCUMENT NUMBER: PREV200200586592
TITLE: Relationship of Helicobacter immunoblot antibody profile to predicted probability of having **diagnosed** idiopathic **parkinsonism**.
AUTHOR(S): Dobbs, S. M. (1); Oxlade, N. (1); Weller, C. (1); Dobbs, J. (1); Charlett, A.
CORPORATE SOURCE: (1) Institute of Psychiatry, London UK
SOURCE: Gut, (September, 2002) Vol. 51, No. Supplement 2, pp. A77. http://gut.bmjjournals.com/. print.
Meeting Info.: XVth International Workshop on Gastrointestinal Pathology and Helicobacter Athens, Greece September 11-14, 2002
ISSN: 0017-5749.
DOCUMENT TYPE: Conference
LANGUAGE: English

L11 ANSWER 5 OF 15 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002047298 MEDLINE
DOCUMENT NUMBER: 21630676 PubMed ID: 11774938
TITLE: Helicobacter **pylori** is not the cause of sudden infant death syndrome (SIDS).
AUTHOR: Ho G Y; Windsor H M; Snowball B; Marshall B J
CORPORATE SOURCE: Department of Microbiology, University of Western Australia, QEII Medical Centre, Nedlands, Australia.
SOURCE: AMERICAN JOURNAL OF GASTROENTEROLOGY, (2001 Dec) 96 (12) 3288-94.
Journal code: 0421030. ISSN: 0002-9270.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020125
Entered Medline: 20020116

AB OBJECTIVES: The cause of **sudden infant death syndrome** (**SIDS**) is unknown, but our previous hypothesis proposed that Helicobacter **pylori** could be a causative organism. In this study, we aimed to test this hypothesis by examining gastric and tracheal tissues from a prospective cohort of **SIDS** infants and re-examining

previously studied paraffin-fixed tissues for H. **pylori**. METHODS: Fresh gastric antral and trachea specimens obtained at postmortem from nine consecutive new cases of **SIDS** in Perth, Western Australia were studied prospectively. Tissues were evaluated for H. **pylori** by rapid urease test (CLOtest), bacterial culture, histology (hematoxylin and eosin, Warthin-Starry Silver, and immmunoperoxidase staining), and polymerase chain reaction (PCR). The latter two tests were also used for the re-examination of paraffin-embedded specimens from infants who died from **SIDS** (n = 17) and other non-**SIDS** causes (n = 7) in Kansas City, Missouri. RESULTS: Specimens from nine consecutive **SIDS** infants in Western Australia showed no evidence of H. **pylori** by any analyses. In the paraffin-embedded gastric and trachea specimens from Missouri, rod and coccoid-shaped bacteria were seen histologically in 33.3% of the specimens, but these were not typical H. **pylori**. Upon analysis by PCR, "H. **pylori** DNA" was **detected** in 53% (9/17) of **SIDS** samples versus 57% (4/7) in non-**SIDS** samples. In all cases the immunoperoxidase stain was negative, suggesting that PCR either 1) gave false positive results in this type of potentially contaminated postmortem specimen or 2) H. **pylori** DNA was indeed present but not increased in prevalence in **SIDS** infants. CONCLUSIONS: H. **pylori** is unlikely to be an etiological agent in **SIDS**.

L11 ANSWER 6 OF 15 MEDLINE
ACCESSION NUMBER: 2002119011 MEDLINE
DOCUMENT NUMBER: 21842594 PubMed ID: 11852823
TITLE: [Helicobacter **pylori**--does it only cause gastroduodenal disease?]. Helicobacter **pylori** w chorobach gornego odcinka przewodu pokarmowego--czy tylko?.
AUTHOR: Wlodarek D; Pakszys W; Barlik M
CORPORATE SOURCE: Zaklad Dietetyki, Katedra Dietetyki i Zywnosci Funkcjonalnej, Wydzial Nauk o Zywieniu Czlowieka i Konsumpcji, SGGW w Warszawie.
SOURCE: POLSKI MERKURIUSZ LEKARSKI, (2001 Nov) 11 (65) 456-9. Ref: 26
Journal code: 9705469. ISSN: 1426-9686.
PUB. COUNTRY: Poland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: Polish
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020221
Last Updated on STN: 20020315
Entered Medline: 20020314

AB Helicobacter **pylori** is a human pathogen that can be found all over the world. It is responsible for the following diseases of gastrointestinal tube: gastritis, gastric ulcer, duodenal ulcer, gastric cancer, gastric lymphomas, Menetier disease. Some research has been done recently trying to identify the connection between H. **pylori** infection and idiopathic **Parkinson**'s Disease morbidity. Some of them show that people with this neurological disease are more likely to have ulcers and also seropositivity in the direction of H. **pylori**. The direct

influence of H. **pylori** infection on **Parkinson**
Disease is not known but the following relations are suggested: H. **pylori** may produce toxins that damage substantia nigra in brain; possible cross reaction of h. **pylori** antibodies with dopaminergic neurons; indirect influence of antacids containing aluminium used to alleviate the symptoms of ulcers. Investigations of the reasons for idiopathic **parkinson** disease draw attention to the influence of food factors. Some researches show that there is a relation between the frequency of eating certain foods and the **parkinson** disease morbidity. We have numerous techniques that allow us to **diagnose** h. **pylori** infection. Those techniques have different sensitivity, accuracy, invasiveness and costs, which **determines** their usefulness in clinical **diagnostics** . Approach to eradication of bacteria is still discussed because H. **pylori** infection doesn't always lead to health problems. Polish Working Group on Helicobacter **pylori**, called by the National Consultant's Team on Gastroenterology explained clearly when eradication is advisable and when it can be waived.

L11 ANSWER 7 OF 15 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-205407 [18] WPIDS
DOC. NO. CPI: C2000-063253
TITLE: Microparticles with adsorbent surface comprising polymer and detergent, used as vaccines, and for targeted delivery of e.g. polypeptides, efficient adsorbance of biologically active macromolecules.
DERWENT CLASS: A14 A23 A26 A96 B04 B07 C03 D16
INVENTOR(S): BARACKMAN, J; KAZZAZ, J; O'HAGEN, D; OTT, G S; SINGH, M
PATENT ASSIGNEE(S): (CHIR) CHIRON CORP
COUNTRY COUNT: 87
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000006123	A1	20000210	(200018)*	EN	59
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9952452	A	20000221	(200029)		
EP 1100468	A1	20010523	(200130)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2002521425	W	20020716	(200261)		73

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000006123	A1	WO 1999-US17308	19990729
AU 9952452	A	AU 1999-52452	19990729
EP 1100468	A1	EP 1999-937664	19990729
		WO 1999-US17308	19990729
JP 2002521425	W	WO 1999-US17308	19990729

JP 2000-561979 19990729

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9952452	A	Based on	WO 200006123
EP 1100468	A1	Based on	WO 200006123
JP 2002521425	W	Based on	WO 200006123

PRIORITY APPLN. INFO: US 1999-285855 19990402; US 1998-124533 19980729

AN 2000-205407 [18] WPIDS

AB WO 200006123 A UPAB: 20000412

NOVELTY - Microparticles with an adsorbent surface are new and comprise:

(1) polymer chosen from poly(alpha -hydroxy acid), polyhydroxy butyric acid, polycaprolactone, polyorthoester, polyanhydride or polycyanoacrylate; and

(2) detergent.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a method of producing microparticles with adsorbent surface to which biologically active macromolecule has been adsorbed.

ACTIVITY - Vaccine; immunomodulating. Microparticle induction of immune response was examined in guinea pigs following intramuscular immunization. Five formulations were tested: (1) PLG/CTAB gp 120 adsorbed (25 mu g); (2) PLG/CTAB gp 120 adsorbed (25 mu g) + aluminum phosphate; (3) soluble gp 120 DNA (25 mu g) + aluminum phosphate; (4) soluble gp 120 DNA (25 mu g) alone; and (5) MF59 protein (50 mg). GMT of serum was as follows: (1) 1,435 plus or minus 383; (2) 3,624 plus or minus 454; (3) 119 plus or minus 606; (4) 101 plus or minus 55; and (5) 3,468 plus or minus 911. Antibody induction (collection and analysis of serum) were performed and geometric mean titer of serum determined.

USE - Used for **diagnosis** or treatment of disease, as vaccines and to raise and immune response. Used to deliver polypeptides, polynucleotides, polynucleosides, antigens, pharmaceuticals, hormones, enzymes, transcription or translation mediators, intermediates in metabolic pathway, immunomodulators or adjuvants including aluminum salts (claimed) such as double- and single stranded sequences including cDNA, prokaryotic or eukaryotic mRNA, genomic RNA and DNA sequences form viral or prokaryotic DNA (RNA and DNA viruses), and synthetic DNA sequences, base analogs of DNA and RNA, antibiotics, antivirals, peptides, oligopeptides, dimers, multimers, antigens derived from bacteria (Bordetella pertussis, Neisseria meningitides (A, B, C, Y), Neisseria gonorrhoeae, Helicobacter **pylori** and/or Haemophilus influenzae), viruses, parasites, fungi and tumors, non-steroidal anti-inflammatory drugs, analgesics, vasodilators, cardiovascular drugs, psychotropics, neuroleptics, antidepressants, anti-**Parkinson** drugs, beta blockers, calcium channel blockers, bradykinin inhibitors, angiotensin-converting enzyme inhibitors, prolactin inhibitors, steroids, hormone antagonists, antihistamines, serotonin antagonists, heparin, chemotherapeutic agents, antineoplastics and growth factors (platelet derived growth factor (PDGF), epithelial growth factor (EGF), KGF, insulin-like growth factor (IGF)-1, IFG-2), FGF, polynucleotides that encode therapeutic or immunogenic proteins, immunogenic proteins and epitopes for use

in vaccines, hormones including peptide hormones (insulin, proinsulin, growth hormone, GHRH, luteinizing hormone releasing hormone (LHRH), EGF, somatostatin, SNX-111, BNP, insulinotropin, ANP, FSH, LH, PSH and hCG), gonadal steroid hormones (androgens, estrogens, progesterone), thyroid-stimulating hormone, inhibin, cholecystokinin, ACTH, CRF, dynorphins, endorphins, endothelin, fibronectin fragments, galanin, gastrin, glucagons, GTP-binding protein fragments, guanylin, leukokinins, magainin, mastoparans, dermaseptin, systemin, neuromedin, neurotensin, pancreastatin, pancreatic polypeptide, substance P, secretin, thymosin, and cytokines (interleukin (IL) 1, IL-2, IL-3, IL-4 and gamma interferon). Used for site-specific targeted delivery.

ADVANTAGE - Efficiently adsorb biologically active macromolecules such as DNA, polypeptides, antigens and adjuvants. Capable of adsorbing wide variety of macromolecules. Flexible delivery systems, particularly for drugs that are highly sensitive and difficult to formulate.

Dwg.0/0

L11 ANSWER 8 OF 15 MEDLINE DUPLICATE 2

ACCESSION NUMBER: 2000492392 MEDLINE
DOCUMENT NUMBER: 20427755 PubMed ID: 10971237
TITLE: Parkinsonism: differential age-trend in Helicobacter **pylori** antibody.
AUTHOR: Dobbs R J; Charlett A; Dobbs S M; Weller C; Peterson D W
CORPORATE SOURCE: Therapeutics in the Elderly, Research Group, Northwick Park and St Mark's Hospitals, Harrow, UK.. dobbs@wellers.demon.co.uk
SOURCE: ALIMENTARY PHARMACOLOGY AND THERAPEUTICS, (2000 Sep) 14 (9) 1199-205.
Journal code: 8707234. ISSN: 0269-2813.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001027
Last Updated on STN: 20001027
Entered Medline: 20001019

AB BACKGROUND: **Parkinsonism** is associated with prodromal peptic ulceration. Dopamine antagonists provoke experimental ulcer, dopaminergic agents protect, and might inhibit growth of Helicobacter **pylori**. OBJECTIVE: To describe the relationship between H. **pylori** serology and **parkinsonism**. METHODS: Serum H. **pylori** anti-urease-IgG antibody was measured in 105 people with (idiopathic) **parkinsonism**, 210 without, from same locality. None had received specific eradication therapy. RESULTS: Controls showed a birth-cohort effect: antibody titre rose from 30 to 90 years (P < 0. 001). **Parkinsonism** obliterated this (disease status. age interaction, P < 0.05), the differential age trend not being attributable to social class. Those with **diagnosed parkinsonism** were more likely to be seropositive (odds ratio 2.04 (95% CI: 1.04, 4.22) P < 0.04) before 72.5 years. Overall, titre fell (P=0.01) by 5 (1, 9)% per unit increase in a global, 30-point rating (median 14 (interquartile range 10.5, 17)) of disease severity. No individual category of

anti-**parkinsonian** medication (92% taking) had a differential lowering effect. CONCLUSIONS: Higher prevalence of seropositivity in **parkinsonism**, before 8th decade, may be due to host susceptibility/reaction, or, conversely, infection with particular H. **pylori** strain(s) lowering dopaminergic status. Absence of a birth cohort effect in **parkinsonism**, despite similar social class representation, may be consequent on eradication, spontaneous (gastric atrophy) or by anti-**parkinsonian** medication.

L11 ANSWER 9 OF 15 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2000499515 MEDLINE
DOCUMENT NUMBER: 20496722 PubMed ID: 11040154
TITLE: An association between sudden infant death syndrome (SIDS) and Helicobacter **pylori** infection.
COMMENT: Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
Comment in: Arch Dis Child. 2001 Jun;84(6):525
AUTHOR: Kerr J R; Al-Khattaf A; Barson A J; Burnie J P
CORPORATE SOURCE: Infectious Diseases Research Group, The University of Manchester, Clinical Sciences Building, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, UK.. jonathankerr@hotmail.com
SOURCE: ARCHIVES OF DISEASE IN CHILDHOOD, (2000 Nov) 83 (5) 429-34.
Journal code: 0372434. ISSN: 1468-2044.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010723
Entered Medline: 20001103

AB BACKGROUND: Helicobacter **pylori** has recently been **detected** in the stomach and trachea of cases of **sudden infant death syndrome** (**SIDS**) and proposed as a cause of **SIDS**. AIMS: To establish the incidence of H **pylori** in the stomach, trachea, and lung of cases of **SIDS** and controls. METHODS: Stomach, trachea, and lung tissues from 32 cases of **SIDS** and eight control cases were examined retrospectively. **Diagnosis** of **SIDS** was based on established criteria. Controls were defined by death within 1 year of age and an identifiable cause of death. Tissues were examined histologically for the presence of bacteria. Extracted DNA from these tissues was tested for H **pylori** ureC and cagA sequences by nested polymerase chain reaction and amplicons **detected** by enzyme linked immunosorbent assay (ELISA). The cut off for each ELISA for each of the tissue types was taken as the mean optical density plus

two times the standard deviation of a range of negative controls. RESULTS: Ages of **SIDS** cases ranged from 2 to 28 weeks. Ages of controls ranged from 3 to 44 weeks. For the ureC gene, 25 **SIDS** cases were positive in one or more tissues compared with one of the controls. For the cagA gene, 25 **SIDS** cases were positive in one or more tissues compared with one of the controls. CONCLUSIONS: There is a highly significant association between H **pylori** ureC and cagA genes in the stomach, trachea, and lung of cases of **SIDS** when compared with controls.

L11 ANSWER 10 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:73545 BIOSIS
DOCUMENT NUMBER: PREV200100073545
TITLE: Insights into the natural history of idiopathic Parkinsonism in relation to Helicobacter **pylori** anti-urease antibody titre.
AUTHOR(S): Dobbs, S. M. (1); Charlett, A.; Dobbs, R. J. (1); Weller, C. (1)
CORPORATE SOURCE: (1) Therapeutics in the Elderly, Northwick Park and St Mark's Hospital, Harrow, HA1 3UJ UK
SOURCE: British Journal of Clinical Pharmacology, (October, 2000) Vol. 50, No. 4, pp. 389. print. Meeting Info.: British Pharmacological Society, Clinical Pharmacology Section Cardiff, Wales, UK July 12-14, 2000 British Pharmacological Society . ISSN: 0306-5251.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L11 ANSWER 11 OF 15 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2001164333 MEDLINE
DOCUMENT NUMBER: 21111175 PubMed ID: 11179988
TITLE: Is sudden infant death syndrome associated with Helicobacter **pylori** infection in children?.
AUTHOR: Elitsur Y; Btriest W; Sabet Z; Neace C; Jiang C; Thomas E
CORPORATE SOURCE: Department of Pediatrics, Marshall University School of Medicine, Huntington, WV 25701-3655, USA.. elitsur@marshall.edu
SOURCE: HELICOBACTER, (2000 Dec) 5 (4) 227-31. Journal code: 9605411. ISSN: 1083-4389.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010625
Last Updated on STN: 20010625
Entered Medline: 20010621
AB Helicobacter **pylori** infection has recently been implicated in the pathogenesis of **sudden infant death syndrome** (**SIDS**). We investigated this association. Twenty-five pairs of gastric and tracheal tissue specimens obtained from autopsies of 25 children with previous **diagnoses** of **SIDS** were available for this study. The presence of H. **pylori** organisms was evaluated by three

different methods: histology (hematoxylin-eosin or Giemsa staining), immunohistochemistry, and nested polymerase chain reaction technique. We were unable to confirm the presence of H. **pylori** organisms by the first two methods. H. **pylori** DNA was identified by nested polymerase chain reaction in six different tissue specimens (stomach, 4; trachea, 2). In no case was H. **pylori** DNA **detected** in both tissues. We concluded that H. **pylori** infection is most likely not associated with **SIDS**.

L11 ANSWER 12 OF 15 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 2000497310 MEDLINE
DOCUMENT NUMBER: 20366366 PubMed ID: 10904422
TITLE: Link between Helicobacter **pylori** infection and idiopathic parkinsonism.
AUTHOR: Dobbs S M; Dobbs R J; Weller C; Charlett A
CORPORATE SOURCE: Therapeutics in the Elderly, Research Group, Northwick Park & St Mark's Hospitals, Harrow, UK.. dobbs@wellers.demon.co.uk
SOURCE: MEDICAL HYPOTHESES, (2000 Aug) 55 (2) 93-8. Journal code: 7505668. ISSN: 0306-9877.
PUB. COUNTRY: SCOTLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001027
Last Updated on STN: 20001027
Entered Medline: 20001013

AB The conventional concept for an environmental cause of idiopathic **parkinsonism** is an insult (e.g. neurotoxin or encephalitis), superimposed on age-related attrition of nigral dopaminergic neurons, and temporally remote from neurological **diagnosis** . To the contrary, we describe the fit of Helicobacter **pylori**. This commonest of known bacterial infections, usually acquired in childhood, persists, and has been linked with peptic ulcer/non-ulcer dyspepsia, immunosuppression and autoimmunity. Acquired immunosuppression, predisposing to auto-immunity, is assessed as a model for the pathogenesis of **parkinsonism** and **parkinsonian**-like attributes of ageing. Eradication of a trigger has potential to change the approach to **parkinsonism**, just as it did to peptic ulcer. The tenet of inevitable age-related attrition of dopaminergic neurons may also require revision.

L11 ANSWER 13 OF 15 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-105663 [09] WPIDS
DOC. NO. CPI: C2000-031695
TITLE: Use of compositions containing a receptor ligand and a receptor ligand binding molecule for treating e.g. infections, inflammatory or immune disease or disorder or cancers.
DERWENT CLASS: B04
INVENTOR(S): BURNS, J M; DEVICO, A L; GALLO, R; LEWIS, G K
PATENT ASSIGNEE(S): (UYMA-N) UNIV MARYLAND BIOTECHNOLOGY INST
COUNTRY COUNT: 87
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9962535	A2	19991209	(200009)*	EN	70
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZA ZW					
AU 9943254	A	19991220	(200021)		
EP 1100527	A2	20010523	(200130)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
US 6399078	B1	20020604	(200242)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9962535	A2		WO 1999-US12137	19990601
AU 9943254	A		AU 1999-43254	19990601
EP 1100527	A2		EP 1999-955219	19990601
			WO 1999-US12137	19990601
US 6399078	B1	Provisional	US 1998-87436P	19980601
			US 1999-323719	19990601

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9943254	A	Based on	WO 9962535
EP 1100527	A2	Based on	WO 9962535

PRIORITY APPLN. INFO: US 1998-87436P 19980601; US 1999-323719 19990601

AN 2000-105663 [09] WPIDS

AB WO 9962535 A UPAB: 20000218

NOVELTY - The use of compositions containing a receptor ligand (RL) and a receptor ligand binding molecule (RLBM) for treating diseases or conditions related to ligand/receptor signaling is new.

DETAILED DESCRIPTION - Method (I) of treating a disease or condition which is caused by or contributed to by the function of a ligand/receptor-mediated signaling pathway or which is dependent upon the extracellular recognition of a receptor by an infectious agent, comprises administering to a patient a composition which includes a RL, and a RLBM, where the composition is capable of antagonizing the function of the receptor or altering the extracellular recognition of the receptor by the infectious agent, to treat the disease or condition.

INDEPENDENT CLAIMS are also included for the following:

(1) a method (II) of inhibiting a chemokine receptor-mediated infection comprising contacting a cell with a formulation which includes a chemokine which binds to the chemokine receptor, and a chemokine binding molecule (CBM) which binds to the chemokine where the formulation is capable of inhibiting the chemokine receptor-mediated infection and suppressing signal transduction from the chemokine receptor; and

(2) a method (III) of treating or preventing infection of a

subject by HIV comprising administering to the subject a composition which includes a chemokine and a CBM, where the composition resulting from the combination of the chemokine and the CBM confers a longer soluble plasma half-life upon the chemokine than the soluble plasma half-life of the chemokine when administered without the CBM and where the composition is further capable of suppressing signal transduction from a receptor to which the chemokine ordinarily binds;

ACTIVITY - Anti-microbial, immunomodulatory, neurotropic, catabolic, etc.

MECHANISM OF ACTION - Chemokine receptor antagonist by competitive inhibition thereby altering the extracellular recognition of the receptor by the infectious agent.

USE - The methods can be used for treating an infectious disease caused by a virus e.g. HIV, Epstein-Barr virus, rhinovirus, poliovirus, rabies virus, reovirus, influenza virus, herpes simplex virus, hepatitis virus, togavirus, varicella-zoster virus, paramyxovirus, cytomegalovirus, subacute sclerosing panencephalitis virus, adenovirus, poxvirus, reovirus, papovavirus, papillmavirus, polyomavirus, slow virus, or bacteria, e.g. Helicobacter **pylori**, Borelia burgdoferi, Legionella pneumophilia, Mycobacterium tuberculosis, M. avium M. intracellulare, M. kansaii, M. gordonae, M. leprae, Staphylococcus aureus, Neisseria gonorrhoeae, N. meningitidis, Listeria monocytogenes, S. pyogenes, S. agalactiae, S. faecalis, S. bovis, S. anginosus, S. pneumoniae, pathogenic Campylobacter species, pathogenic Enterococc us species, Harmophilus influenzae, Bacillus antracis, Corynebacterium diphtheriae, Enterobacter aerogenes, Klebsiella pneumoniae, pasturella multocide, pathogenic Bacteroides fragilis group species, Fusobacterium nucleatum, Streptobacillus moniliformis, treponema pallidium, Treponema pertenue, Leptospira, and Actimomyces isrealli, fungi, e.g. Cryptocossuc neoformans, Histoplasma capsulatum, Coccidioides immitis, Blastomyces dermatidis, Chlamydia trachomatis, and Candida albicans, or a microbe, e.g. Bacillus anthracis, a pathogenic Bordetella species, Bordetella pertussis, Clostridium botulinum, C. tetani, Vibrio cholerae, Corynebactreium diphtheriae, E. coli, Pseudomonase aeruginosa, and Shigella dysenteriae (claimed). They can also be used for treating an inflammatory or an immune disease or disorder (e.g. AIDS) or cancer (claimed). In particular, they can be used for treating e.g. systemic lupus erythematosus, glomerulonephritis, vasculitis, pyogenic infections, immune complex disease, adult respiratory distress syndrome, septic shock or multiple organ failure, vascular diseases or disorders, cardiac disorders, cardiovascular system diseases and disorders, wound healing, limb regeneration, periodontal regeneration, neurological damage or diseases, e.g., Alzheimer's disease, **Parkinson**'s disease, AIDS-related complex, cerebral palsy, depression or neuroendocrine disorders such as hyperthyroidism or hypertension, other diseases, conditions or disorders which result from aberrations or alterations of cell receptor-dependent processes including collateral growth and remodeling of cardiac blood vessels, angiogenesis, cellular transformation through autocrine or paracrine mechanisms, chemotactic stimulation of cells (e.g. endothelial), neurite outgrowth of neuronal precursor cell types (e.g. PC12 phaeochromoctoma). They can also be used for treating e.g. insulin-dependent hypoglycemic condition or amyloid diseases ad to promote skeletal muscle development thereby increasing muscle mass in livestock and obviating the need for excessive use of antibiotics

and hormones to improve feed conversion and weight gain in animals. The methods can also be used in drug **screening**.

ADVANTAGE - The combination of the RL and the RLBM has a longer plasma half-life than the RL alone and provides more effective therapy. Since the complexes are unable to trigger receptors, they should prove to be free from undesirable side effects resulting from the continued activation of their target receptor as has been observed in the use of chemokines to block HIV infection.

Dwg.0/7

L11 ANSWER 14 OF 15 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 1997-281148 [25] WPIDS
DOC. NO. NON-CPI: N1997-232886
DOC. NO. CPI: C1997-090433
TITLE: Identification and prevention of **sudden infant death syndrome** - by **detection** and treatment of Helicobacter **pylori** infection in the infant's mother or a person who comes into contact with the infant.
DERWENT CLASS: B04 B05 D16 S03
INVENTOR(S): HEDNER, J; PETTERSSON, A
PATENT ASSIGNEE(S): (HEDN-I) HEDNER J; (PETT-I) PETTERSSON A
COUNTRY COUNT: 20
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9717612	A1	19970515	(199725)*	EN	15
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: JP US					
JP 2000500859	W	20000125	(200016)		17
EP 1019726	A1	20000719	(200036)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
US 6083756	A	20000704	(200036)		
EP 1121938	A2	20010808	(200146)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
EP 1019726	B1	20020213	(200212)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
DE 69619283	E	20020321	(200227)		
ES 2173326	T3	20021016	(200279)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9717612	A1		WO 1996-SE1428	19961106
JP 2000500859	W		WO 1996-SE1428	19961106
			JP 1997-518127	19961106
EP 1019726	A1		EP 1996-938587	19961106
			WO 1996-SE1428	19961106
US 6083756	A		WO 1996-SE1428	19961106
			US 1998-68363	19980507
EP 1121938	A2	Div ex	EP 1996-938587	19961106
			EP 2001-108549	19961106
EP 1019726	B1		EP 1996-938587	19961106
			WO 1996-SE1428	19961106
		Related to	EP 2001-108549	19961106

Searcher : Shears 308-4994

DE 69619283	E	DE 1996-619283	19961106
		EP 1996-938587	19961106
		WO 1996-SE1428	19961106
ES 2173326	T3	EP 1996-938587	19961106

FILING DETAILS:

PATENT NO	KIND		PATENT NO
JP 2000500859	W	Based on	WO 9717612
EP 1019726	A1	Based on	WO 9717612
US 6083756	A	Based on	WO 9717612
EP 1121938	A2	Div ex	EP 1019726
EP 1019726	B1	Related to	EP 1121938
		Based on	WO 9717612
DE 69619283	E	Based on	EP 1019726
		Based on	WO 9717612
ES 2173326	T3	Based on	EP 1019726

PRIORITY APPLN. INFO: SE 1995-3937 19951107
AN 1997-281148 [25] WPIDS
AB WO 9717612 A UPAB: 19970619
Identification of an infant, born or unborn, being particularly susceptible to **sudden infant death syndrome** (**SIDS**), comprises **determination** of a Helicobacter **pylori** (HP) infection in the infant's mother, a close relative or a person expected to come into close bodily contact with the infant.
USE - The methods can be used for identifying infants susceptible to SIDS and for preventing SIDS (claimed).
Dwg.0/0

L11 ANSWER 15 OF 15 MEDLINE
ACCESSION NUMBER: 96133400 MEDLINE
DOCUMENT NUMBER: 96133400 PubMed ID: 8536488
TITLE: Gastroesophageal reflux in childhood.
AUTHOR: Fonkalsrud E W; Ament M E
CORPORATE SOURCE: Pediatric Surgery, UCLA School of Medicine, USA.
SOURCE: CURRENT PROBLEMS IN SURGERY, (1996 Jan) 33 (1) 1-70.
Ref: 275
Journal code: 0372617. ISSN: 0011-3840.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199602
ENTRY DATE: Entered STN: 19960221
Last Updated on STN: 19960221
Entered Medline: 19960207
AB Gastroesophageal reflux (GER) is one of the most frequent symptomatic clinical disorders affecting the gastrointestinal tract of infants and children. During the past 2 decades, GER has been recognized more frequently because of an increased awareness of the condition and also because of the more sophisticated **diagnostic** techniques that have been developed for both identifying and quantifying the disorder. Gastroesophageal

fundoplication is currently one of the three most common major operations performed on infants and children by pediatric surgeons in the United States. Normal gastroesophageal function is a complex mechanism that depends on effective esophageal motility, timely relaxation and contractility of the lower esophageal sphincter, the mean intraluminal pressure in the stomach, the effectiveness of contractility in emptying of the stomach, and the ease of gastric outflow. More than one of these factors are often abnormal in the same child with symptomatic GER. In addition, in patients with GER disease, and particularly in those patients with neurologic disorders, there appears to be a high prevalence of autonomic neuropathy in which esophagogastric transit and gastric emptying are frequently delayed, producing a somewhat complex foregut motility disorder. GER has a different course and prognosis depending on the age of onset. The incompetent lower esophageal sphincter mechanism present in most newborn infants combined with the increased intraabdominal pressure from crying or straining commonly becomes much less frequent as a cause of vomiting after the age of 4 months. Chalasia and rumination of infancy are self-limited and should be carefully separated from symptomatic GER, which requires treatment. The most frequent complications of recurrent GER in childhood are failure to thrive as a result of caloric deprivation and recurrent bronchitis or pneumonia caused by repeated pulmonary aspiration of gastric fluid. Children with GER disease commonly have more refluxing episodes when in the supine position, particularly during sleep. The reflux of acid into the mid or upper esophagus may stimulate vagal reflexes and produce reflex laryngospasm, bronchospasm, or both, which may accentuate the symptoms of asthma. Reflux may also be a cause of obstructive apnea in infants and possibly a cause of recurrent stridor, acute hypoxia, and even the **sudden infant death syndrome**.
Premature infants with respiratory distress syndrome have a high incidence of GER. Esophagitis and severe dental carries are common manifestations of GER in childhood. Barrett's columnar mucosal changes in the lower esophagus are not infrequent in adolescent children with chronic GER, particularly when Heliobacter **pylori** is present in the gastric mucosa. Associated disorders include esophageal dysmotility, which has been recognized in approximately one third of children with severe GER. Symptomatic GER is estimated to occur in 30% to 80% of infants who have undergone repair of esophageal atresia malformations. Neurologically impaired children are at high risk for having symptomatic GER, particularly if nasogastric or gastrostomy feedings are necessary. Delayed gastric emptying (DGE) has been documented with increasing frequency in infants and children who have symptoms of GER, particularly those with neurologic disorders. DGE may also be a cause of gas bloat, gagging, and breakdown or slippage of a well-constructed gastroesophageal fundoplication. The most helpful test for **diagnosing** and quantifying GER in childhood is the 24-hour esophageal pH monitoring study. Miniaturized probes that are small enough to use easily in the newborn infant are available. This study is 100% accurate in **diagnosing** reflux when the esophageal pH is less than 4.0 for more than 5% of the total monitored time.

L1 17728 SEA FILE=HCAPLUS ABB=ON PLU=ON ADD(10A)(ATTENTION DEFICIT) OR ADHD OR ATTENTION(3W)DISORDER OR AUTISM OR

PARKINSON? OR PDD OR PERVAS? DEVELOP? DISORDER OR DYSAUTONOM? OR DYS AUTONOM? OR SIDS OR SUDDEN INFANT DEATH SYNDROME OR AUTISTIC

L2 9 SEA FILE=HCAPLUS ABB=ON PLU=ON L1 AND PYLORI
L3 191 SEA L2
L4 5 SEA L3 AND (STOOL OR FECES OR FAECES OR FECAL OR FAECAL)

L5 23 SEA L3 AND (IMMUNOASSAY? OR ASSAY?)
L6 61 SEA L3 AND (DETERM? OR DETECT? OR DET## OR SCREEN? OR DIAGNOS?)
L7 74 SEA L4 OR L5 OR L6
L12 3 SEA L7 AND ANTIGEN

L13 1 L12 NOT L10

L13 ANSWER 1 OF 1 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-639204 [73] WPIDS
DOC. NO. NON-CPI: N2001-477779
DOC. NO. CPI: C2001-189094
TITLE: Use of activated protein C or compound with activated protein C activity for treating a disease or condition, e.g. cancers.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): CIACCIA, A V; GELBERT, L M; GRINNELL, B W; JONES, B E; JOYCE, D E
PATENT ASSIGNEE(S): (ELIL) LILLY & CO ELI
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001072328	A2	20011004	(200173)*	EN	38

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2001045319	A	20011008	(200208)		
EP 1267915	A2	20030102	(200310)	EN	

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001072328	A2	WO 2001-US5823	20010321
AU 2001045319	A	AU 2001-45319	20010321
EP 1267915	A2	EP 2001-918217	20010321
		WO 2001-US5823	20010321

FILING DETAILS:

PATENT NO	KIND	PATENT NO

AU 2001045319 A Based on WO 200172328
EP 1267915 A2 Based on WO 200172328

PRIORITY APPLN. INFO: US 2000-192755P 20000328
AN 2001-639204 [73] WPIDS
AB WO 200172328 A UPAB: 20011211
NOVELTY - Use of an activated protein C (I) or a compound (II) having activated protein C activity for treating a disease or pathological condition in a patient, by direct regulation of the expression of specific genes associated with the disease or pathological condition.

DETAILED DESCRIPTION - Treating a patient suffering from a disease or pathological condition associated with apoptotic cell death; increasing the activity of Bcl-2 or human IAP homolog B in cell affected by a disease or pathological condition associated with apoptosis; treating a patient suffering from a disease or pathological condition where tumor necrosis factor (TNF)- alpha is a primary modulator of pathophysiology or cell-cell adhesion is a modulator of pathophysiology; increasing angiogenesis in a patient in need of wound healing or tissue repair where proliferating cell nuclear **antigen** (PCNA) or Gu protein is a regulator of cell growth and survival; or treating a patient suffering from a disease or pathological condition induced by nuclear factor kappa B (NF-kappaB); comprises administering (I) or (II) to the patient.

INDEPENDENT CLAIMS are also included for the following:

(1) use of (I) in the manufacture of a medicament for the treatment of a disease or pathological condition associated with above said conditions;

(2) **screening** to identify test substances which induce or repress expression of genes which are induced or repressed by (I), by contacting a cell with a test substance, monitoring expression of a transcript or its translation product, where the transcript specifically hybridizes to one or more genes selected from first and second group of molecules that are given in the specification, where a test substance is identified if it increases expression of a transcript which specifically hybridizes to one or more genes in the first group and decreases expression of a transcript which specifically hybridizes to one or more genes in the second group; and

(3) **screening** to identify test substances which modulate the activity of (I) on the induction or repression of genes, by contacting a cell with a test substance in combination with (I), monitoring expression of a transcript or its translation product, where the transcript specifically hybridizes to one or more genes selected from first and second group of molecules that are given in the specification, where a test substance in combination with (I) is identified if it increases expression of a transcript which specifically hybridizes to one or more genes in the first group where the increase being greater than with (I) alone and decreases expression of a transcript which specifically hybridizes to one or more genes in the second group where the decrease being greater than (I) alone.

ACTIVITY - Antirheumatic; antiarthritic; vasotropic; antidiabetic; neuroprotective; nootropic; antiulcer; cardiant; antiparkinsonian; anti-HIV; cytostatic; antibacterial; analgesic; antiinflammatory; neuroprotective; antipsoriatic; antithyroid; immunosuppressive; thyromimetic; dermatological; nephrotropic; virucide; hepatotropic; osteopathic; antiarteriosclerotic;

tranquilizer; antianemic; anticonvulsant; neuroleptic; fungicide; protozoacide; antiasthmatic; antiallergic; antidepressant; antimanic; antianginal; hypotensive.

No supporting data given.

MECHANISM OF ACTION - Repressor of the transcription of endothelial leukocyte adhesion molecule-1 (ELAM-1), vascular cell adhesion molecule-1 (VCAM-1), PECAM-1 and human CX3C chemokines precursor; apoptosis inhibitor (claimed).

The inhibition of apoptosis by recombinant activated protein C (rhAPC) in primary human umbilical venous endothelial cells (HUVEC) or the immortalized endothelial cell line (Eahy926) was **determined** by utilizing the APOPercentage Apoptosis **Assay**. Briefly, adherent cells (HUVEC, Eahy926, or 293) were seeded at 3 multiply 104 cells per well and treated with 1 micro g/ml/hour staurosporine (an alkaloid isolated from the culture broth of Streptomyces staurospores, and a potent inhibitor of protein kinase C and inducer of apoptosis), or with staurosporine and rhAPC (pretreatment 16 hours). Cell were prepared and stained. Significant inhibition of apoptosis by rhAPC was observed in both the HUVEC and Eahy926 endothelial cell lines.

USE - (I) or (II) is useful for the disease or pathological condition including rheumatoid arthritis, inflammatory bowel disease, vasculitis, renal ischemia, insulin-dependent diabetes mellitus, pancreatitis, psoriasis, multiple sclerosis, Hashimoto's thyroiditis, Grave's disease, transplant rejection, systemic lupus erythematosus, autoimmune gastritis, fibrosing lung disease, HIV-induced lymphoma, fulminant viral hepatitis B, fulminant viral hepatitis C, chronic hepatitis, chronic cirrhosis, Helicobacter **pylori**-associated ulceration, cytoprotection during cancer treatment, adjuvant to chemotherapy, chronic glomerulonephritis, osteoporosis, aplastic anemia, myelodysplasia, neurodegenerative diseases such as Alzheimer's disease and **Parkinson**'s disease, glutamate-induced neurotoxicity, Crohn's disease, ulcerating colitis, arthritis, acute peritoneal inflammation and heart failure, neuronal degeneration diseases, graft versus host reactions, acute inflammatory conditions, systemic inflammatory responses, acute phase response, ischemic reperfusion injury, atherosclerosis, HIV infection and cancer (claimed).

(I) or (II) is also useful for treating coronary artery atherosclerosis, arterial restenosis following balloon angioplasty, hypertension, coronary disease after transplantation, pregnancy-induced hypertension and pre-eclampsia, bacterial, fungal, protozoan and viral infections (particularly injections caused by HIV-1 or HIV-2), pain, anorexia, bulimia, asthma, hypotension, urinary retention, angina pectoris, allergies, and psychotic and neuronal disorders including anxiety, schizophrenia, manic depression, delirium, dementia, severe mental retardation and dyskinesias such as Huntington's disease or Gilles dela Tourette's syndrome.

Dwg.0/2

FILE 'MEDLINE' ENTERED AT 12:11:00 ON 21 FEB 2003)

L14 6383 SEA FILE=MEDLINE ABB=ON PLU=ON "AUTISTIC DISORDER"/CT

L15 21573 SEA FILE=MEDLINE ABB=ON PLU=ON "PARKINSON DISEASE"/CT

L16 299 SEA FILE=MEDLINE ABB=ON PLU=ON "ATTENTION DEFICIT AND DISRUPTIVE BEHAVIOR DISORDERS"/CT

L17 6964 SEA FILE=MEDLINE ABB=ON PLU=ON "ATTENTION DEFICIT DISORDER WITH HYPERACTIVITY"/CT

Searcher : Shears 308-4994

L18 5072 SEA FILE=MEDLINE ABB=ON PLU=ON "SUDDEN INFANT DEATH"/CT

L19 14068 SEA FILE=MEDLINE ABB=ON PLU=ON "HELICOBACTER PYLORI"/CT

L20 31 SEA FILE=MEDLINE ABB=ON PLU=ON (L14 OR L15 OR L16 OR L17 OR L18) AND L19

L20 ANSWER 1 OF 31 MEDLINE
AN 2002351416 MEDLINE
TI Helicobacter pylori and SIDS: the jury is in at last!.
AU Elitsur Yoram
SO AMERICAN JOURNAL OF GASTROENTEROLOGY, (2002 Jun) 97 (6) 1576-7.
Journal code: 0421030. ISSN: 0002-9270.

L20 ANSWER 2 OF 31 MEDLINE
AN 2002304568 MEDLINE
TI Is there an infectious component behind headaches and SIDS?.
AU Das Pam
SO LANCET, (2002 May 4) 359 (9317) 1584.
Journal code: 2985213R. ISSN: 0140-6736.

L20 ANSWER 3 OF 31 MEDLINE
AN 2002271366 MEDLINE
TI Autism and gastrointestinal symptoms.
AU Horvath Karoly; Perman Jay A
SO CURRENT GASTROENTEROLOGY REPORTS, (2002 Jun) 4 (3) 251-8. Ref: 33
Journal code: 100888896. ISSN: 1522-8037.
AB Autism is a collection of behavioral symptoms characterized by dysfunction in social interaction and communication in affected children. It is typically associated with restrictive, repetitive, and stereotypic behavior and manifests within the first 3 years of life. The cause of this disorder is not known. Over the past decade, a significant upswing in research has occurred to examine the biologic basis of autism. Recent clinical studies have revealed a high prevalence of gastrointestinal symptoms, inflammation, and dysfunction in children with autism. Mild to moderate degrees of inflammation were found in both the upper and lower intestinal tract. In addition, decreased sulfation capacity of the liver, pathologic intestinal permeability, increased secretory response to intravenous secretin injection, and decreased digestive enzyme activities were reported in many children with autism. Treatment of digestive problems appears to have positive effects on autistic behavior. These new observations represent only a piece of the unsolved autism "puzzle" and should stimulate more research into the brain-gut connection.

L20 ANSWER 4 OF 31 MEDLINE
AN 2002119011 MEDLINE
TI [Helicobacter pylori--does it only cause gastroduodenal disease?]. Helicobacter pylori w chorobach gornego odcinka przewodu pokarmowego--czy tylko?.
AU Wlodarek D; Pakszys W; Barlik M
SO POLSKI MERKURIUSZ LEKARSKI, (2001 Nov) 11 (65) 456-9. Ref: 26
Journal code: 9705469. ISSN: 1426-9686.
AB Helicobacter pylori is a human pathogen that can be found all over the world. It is responsible for the following diseases of gastrointestinal tube: gastritis, gastric ulcer, duodenal ulcer, gastric cancer, gastric lymphomas, Menetier disease. Some research

has been done recently trying to identify the connection between H. pylori infection and idiopathic Parkinson's Disease morbidity. Some of them show that people with this neurological disease are more likely to have ulcers and also seropositivity in the direction of H. pylori. The direct influence of H. pylori infection on Parkinson Disease is not known but the following relations are suggested: H. pylori may produce toxins that damage substantia nigra in brain; possible cross reaction of h. pylori antibodies with dopaminergic neurons; indirect influence of antacids containing aluminium used to alleviate the symptoms of ulcers. Investigations of the reasons for idiopathic parkinson disease draw attention to the influence of food factors. Some researches show that there is a relation between the frequency of eating certain foods and the parkinson disease morbidity. We have numerous techniques that allow us to diagnose h. pylori infection. Those techniques have different sensitivity, accuracy, invasiveness and costs, which determines their usefulness in clinical diagnostics. Approach to eradication of bacteria is still discussed because H. pylori infection doesn't always lead to health problems. Polish Working Group on Helicobacter pylori, called by the National Consultant's Team on Gastroenterology explained clearly when eradication is advisable and when it can be waived.

L20 ANSWER 5 OF 31 MEDLINE
AN 2002047298 MEDLINE
TI Helicobacter pylori is not the cause of sudden infant death syndrome (SIDS).
AU Ho G Y; Windsor H M; Snowball B; Marshall B J
SO AMERICAN JOURNAL OF GASTROENTEROLOGY, (2001 Dec) 96 (12) 3288-94. Journal code: 0421030. ISSN: 0002-9270.
AB OBJECTIVES: The cause of sudden infant death syndrome (SIDS) is unknown, but our previous hypothesis proposed that Helicobacter pylori could be a causative organism. In this study, we aimed to test this hypothesis by examining gastric and tracheal tissues from a prospective cohort of SIDS infants and re-examining previously studied paraffin-fixed tissues for H. pylori. METHODS: Fresh gastric antral and trachea specimens obtained at postmortem from nine consecutive new cases of SIDS in Perth, Western Australia were studied prospectively. Tissues were evaluated for H. pylori by rapid urease test (CLOtest), bacterial culture, histology (hematoxylin and eosin, Warthin-Starry Silver, and immmunoperoxidase staining), and polymerase chain reaction (PCR). The latter two tests were also used for the re-examination of paraffin-embedded specimens from infants who died from SIDS (n = 17) and other non-SIDS causes (n = 7) in Kansas City, Missouri. RESULTS: Specimens from nine consecutive SIDS infants in Western Australia showed no evidence of H. pylori by any analyses. In the paraffin-embedded gastric and trachea specimens from Missouri, rod and coccoid-shaped bacteria were seen histologically in 33.3% of the specimens, but these were not typical H. pylori. Upon analysis by PCR, "H. pylori DNA" was detected in 53% (9/17) of SIDS samples versus 57% (4/7) in non-SIDS samples. In all cases the immunoperoxidase stain was negative, suggesting that PCR either 1) gave false positive results in this type of potentially contaminated postmortem specimen or 2) H. pylori DNA was indeed present but not increased in prevalence in SIDS infants. CONCLUSIONS: H. pylori is unlikely to be an etiological agent in SIDS.

L20 ANSWER 6 OF 31 MEDLINE

AN 2001689703 MEDLINE
TI Sudden infant death syndrome and enteric infection.
AU Reid G M
SO MEDICAL HYPOTHESES, (2001 Nov) 57 (5) 580-2.
Journal code: 7505668. ISSN: 0306-9877.
AB The association of Helicobacter pylori in the stomach, trachea and lungs with the incidence of SIDS, gastric ulcers and cancer may have a counterpart in animals. In field studies of white muscle disease (WMD) and hepatic necrosis in selenium-deficient pigs dying suddenly, veterinarians identified gastric ulcers in 40% of inspected piglets. The lesion was also commonly observed by researchers in experimentally produced vitamin E-selenium deficiency and other researchers suspected that gastric ulcers in swine may be associated with vitamin E-selenium deficiency. Mice preferentially concentrated (75)selenium in peritoneal exudative cells (PEC) when (75)selenium as selenium selenate was administered by stomach tube to selenium-deficient mice. Selenium concentrated in PECs as glutathione peroxidase (GSHP(x)). GSHP(x)-deficient leucocytes in peritoneal exudate failed to kill yeast cells. GSHP(x) deficiency has also been associated with decreased microbicidal activity of leucocytes in patients with chronic granulomatosis. The selenium-deficient swine were usually growing rapidly in crowded conditions, and, apart from WMD and hepatic necrosis, edema was prominent in the spiral colon, subcutaneous tissues, lungs and submucosa of the stomach. The elevated immunological response in the spleen and lungs of SIDS victims suggests an initial defective microbicidal propensity of the peritoneal exudative cells. Copyright 2001 Harcourt Publishers Ltd.

L20 ANSWER 7 OF 31 MEDLINE
AN 2001678797 MEDLINE
TI Current controversies associated with Helicobacter pylori infection in the pediatric population.
AU Sherman P M; Macarthur C
SO FRONTIERS IN BIOSCIENCE, (2001 Dec 1) 6 E187-92. Ref: 65
Journal code: 9702166. ISSN: 1093-4715.
AB Helicobacter pylori is a human bacterial gastric pathogen, fulfilling each of Koch's postulates for causal inference for ulceration in children and adults. In addition many reports purport to show that the organism causes a variety of extra-intestinal manifestations in children. This review of the English language literature provides evidence that H. pylori is likely a cause of unexplained iron deficiency (sideropenic) anemia in children, even in the absence of gastrointestinal bleeding. Much stronger evidence is required however, before H. pylori infection can be considered as an etiologic agent in recurrent abdominal pain of childhood, unexplained short stature, protracted diarrhea in pre-schoolers and sudden infant death syndrome.

L20 ANSWER 8 OF 31 MEDLINE
AN 2001440645 MEDLINE
TI Reduced L-dopa absorption and increased clinical fluctuations in Helicobacter pylori-infected Parkinson's disease patients.
AU Pierantozzi M; Pietroiusti A; Sancesario G; Lunardi G; Fedele E; Giacomini P; Frasca S; Galante A; Marciani M G; Stanzione P
SO NEUROLOGICAL SCIENCES, (2001 Feb) 22 (1) 89-91.
Journal code: 100959175. ISSN: 1590-1874.
AB We report that the area under the curve of L-dopa plasma

concentration, following the administration of a single 250 mg L-dopa dose, is augmented after Helicobacter pylori (HP) eradication in six Parkinson's disease (PD) patients showing high IgG antibody titer against HP. A prolongation of L-dopa clinical benefit was also observed. We suggest that HP infection-activated gastric alterations may be responsible, at least in part, for the reported erratic efficacy of oral L-dopa therapy in some advanced PD patients. Given the high percentage of HP-positivity in the age cohorts including the largest prevalence of PD patients, we propose that HP eradication be recommended in all PD patients under L-dopa therapy.

L20 ANSWER 9 OF 31 MEDLINE
AN 2001276606 MEDLINE
TI No association in a Chinese population.
AU Leung W K; Yu J; To K F
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 10 OF 31 MEDLINE
AN 2001276605 MEDLINE
TI Controls not matched.
AU Marshall B J; Ho G Y
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 11 OF 31 MEDLINE
AN 2001276604 MEDLINE
TI The need for further evidence for the proposed role of Helicobacter pylori in SIDS.
AU Blackwell C C; Weir D M; Busuttil A
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 12 OF 31 MEDLINE
AN 2001276603 MEDLINE
TI H pylori DNA may not imply infection.
AU Doherty C P; Mackay W G; Weaver L T; Shepherd A J; Williams C L
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 13 OF 31 MEDLINE
AN 2001276602 MEDLINE
TI Dwelling crowding as a pertinent factor.
AU Beggs P J
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 14 OF 31 MEDLINE
AN 2001276601 MEDLINE
TI Death kisses for newborns?.
AU Vieth M; Stolte M; De Groote D; Deeg K H; Seitz G
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 15 OF 31 MEDLINE
AN 2001276600 MEDLINE
TI Association is not the same as causation.
AU Richardson M

SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 16 OF 31 MEDLINE
AN 2001276599 MEDLINE
TI Control your controls and conclusions.
AU Koletzko S; Konstantopoulos N; Lehn N; Forman D
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 17 OF 31 MEDLINE
AN 2001276598 MEDLINE
TI Ammonia--not the culprit.
AU Wiklund L; Ronquist G; George M
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 18 OF 31 MEDLINE
AN 2001276597 MEDLINE
TI Association between SIDS and H pylori infection.
AU Franciosi R A
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 19 OF 31 MEDLINE
AN 2001276596 MEDLINE
TI Helicobacter pylori.
AU Murphy M S
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2001 Jun) 84 (6) 525.
Journal code: 0372434. ISSN: 1468-2044.

L20 ANSWER 20 OF 31 MEDLINE
AN 2001164333 MEDLINE
TI Is sudden infant death syndrome associated with Helicobacter pylori infection in children?.
AU Elitsur Y; Btriest W; Sabet Z; Neace C; Jiang C; Thomas E
SO HELICOBACTER, (2000 Dec) 5 (4) 227-31.
Journal code: 9605411. ISSN: 1083-4389.
AB Helicobacter pylori infection has recently been implicated in the pathogenesis of sudden infant death syndrome (SIDS). We investigated this association. Twenty-five pairs of gastric and tracheal tissue specimens obtained from autopsies of 25 children with previous diagnoses of SIDS were available for this study. The presence of H. pylori organisms was evaluated by three different methods: histology (hematoxylin-eosin or Giemsa staining), immunohistochemistry, and nested polymerase chain reaction technique. We were unable to confirm the presence of H. pylori organisms by the first two methods. H. pylori DNA was identified by nested polymerase chain reaction in six different tissue specimens (stomach, 4; trachea, 2). In no case was H. pylori DNA detected in both tissues. We concluded that H. pylori infection is most likely not associated with SIDS.

L20 ANSWER 21 OF 31 MEDLINE
AN 2001126802 MEDLINE
TI Helicobacter pylori and sudden-infant-death syndrome.
AU Rowland M; Drumm B
SO LANCET, (2001 Feb 3) 357 (9253) 327.
Journal code: 2985213R. ISSN: 0140-6736.

L20 ANSWER 22 OF 31 MEDLINE
AN 2000499515 MEDLINE
TI An association between sudden infant death syndrome (SIDS) and Helicobacter pylori infection.
AU Kerr J R; Al-Khattaf A; Barson A J; Burnie J P
SO ARCHIVES OF DISEASE IN CHILDHOOD, (2000 Nov) 83 (5) 429-34.
Journal code: 0372434. ISSN: 1468-2044.
AB BACKGROUND: Helicobacter pylori has recently been detected in the stomach and trachea of cases of sudden infant death syndrome (SIDS) and proposed as a cause of SIDS. AIMS: To establish the incidence of H pylori in the stomach, trachea, and lung of cases of SIDS and controls. METHODS: Stomach, trachea, and lung tissues from 32 cases of SIDS and eight control cases were examined retrospectively. Diagnosis of SIDS was based on established criteria. Controls were defined by death within 1 year of age and an identifiable cause of death. Tissues were examined histologically for the presence of bacteria. Extracted DNA from these tissues was tested for H pylori ureC and cagA sequences by nested polymerase chain reaction and amplicons detected by enzyme linked immunosorbent assay (ELISA). The cut off for each ELISA for each of the tissue types was taken as the mean optical density plus two times the standard deviation of a range of negative controls. RESULTS: Ages of SIDS cases ranged from 2 to 28 weeks. Ages of controls ranged from 3 to 44 weeks. For the ureC gene, 25 SIDS cases were positive in one or more tissues compared with one of the controls. For the cagA gene, 25 SIDS cases were positive in one or more tissues compared with one of the controls. CONCLUSIONS: There is a highly significant association between H pylori ureC and cagA genes in the stomach, trachea, and lung of cases of SIDS when compared with controls.

L20 ANSWER 23 OF 31 MEDLINE
AN 2000492392 MEDLINE
TI Parkinsonism: differential age-trend in Helicobacter pylori antibody.
AU Dobbs R J; Charlett A; Dobbs S M; Weller C; Peterson D W
SO ALIMENTARY PHARMACOLOGY AND THERAPEUTICS, (2000 Sep) 14 (9) 1199-205.
Journal code: 8707234. ISSN: 0269-2813.
AB BACKGROUND: Parkinsonism is associated with prodromal peptic ulceration. Dopamine antagonists provoke experimental ulcer, dopaminergic agents protect, and might inhibit growth of Helicobacter pylori. OBJECTIVE: To describe the relationship between H. pylori serology and parkinsonism. METHODS: Serum H. pylori anti-urease-IgG antibody was measured in 105 people with (idiopathic) parkinsonism, 210 without, from same locality. None had received specific eradication therapy. RESULTS: Controls showed a birth-cohort effect: antibody titre rose from 30 to 90 years ($P < 0.001$). Parkinsonism obliterated this (disease status. age interaction, $P < 0.05$), the differential age trend not being attributable to social class. Those with diagnosed parkinsonism were more likely to be seropositive (odds ratio 2.04 (95% CI: 1.04, 4.22) $P < 0.04$) before 72.5 years. Overall, titre fell (P=0.01) by 5 (1, 9)% per unit increase in a global, 30-point rating (median 14 (interquartile range 10.5, 17)) of disease severity. No individual category of anti-parkinsonian medication (92% taking) had a differential lowering effect. CONCLUSIONS: Higher prevalence of seropositivity in parkinsonism, before 8th decade, may be due to

host susceptibility/reaction, or, conversely, infection with particular H. pylori strain(s) lowering dopaminergic status. Absence of a birth cohort effect in parkinsonism, despite similar social class representation, may be consequent on eradication, spontaneous (gastric atrophy) or by anti-parkinsonian medication.

L20 ANSWER 24 OF 31 MEDLINE
AN 1999369060 MEDLINE
TI Association of Helicobacter pylori infection and Parkinson's disease already proposed.
AU Altschuler E L
SO ACTA NEUROLOGICA SCANDINAVICA, (1999 Aug) 100 (2) 122.
Journal code: 0370336. ISSN: 0001-6314.

L20 ANSWER 25 OF 31 MEDLINE
AN 1999169496 MEDLINE
TI Sudden infant death syndrome, long QT interval, and Helicobacter pylori.
AU Kerr J R
SO JOURNAL OF CLINICAL PATHOLOGY, (1998 Dec) 51 (12) 943-4.
Journal code: 0376601. ISSN: 0021-9746.

L20 ANSWER 26 OF 31 MEDLINE
AN 1999122644 MEDLINE
TI Parkinsonism: siblings share Helicobacter pylori seropositivity and facets of syndrome.
AU Charlett A; Dobbs R J; Dobbs S M; Weller C; Brady P; Peterson D W
SO ACTA NEUROLOGICA SCANDINAVICA, (1999 Jan) 99 (1) 26-35.
Journal code: 0370336. ISSN: 0001-6314.
AB OBJECTIVE: Given a history of peptic ulcer is more frequent in parkinsonism, to investigate the role of Helicobacter pylori in its pathogenesis and of cross-infection in familial aggregation. METHODS: Facets of parkinsonism were quantified in 33 elderly subjects with idiopathic parkinsonism and in their 39 siblings with double the number of controls, all obeying inclusion/exclusion criteria. Specific-IgG antibody was assayed. RESULTS: Siblings, compared with controls, had brady/hypokinesia of gait (P< or =0.002), bradykinesia of hands (P = 0.01), abnormal posture (P = 0.001), rigidity (P < 0.001) and seborrhoea/seborrhoeic dermatitis (P = 0.02). Both parkinsonians and siblings differed from controls in the odds of being H. pylori seropositive [odds ratios 3.04 (95% C.I.: 1.22, 7.63) and 2.94 (1.26, 6.86) respectively, P < 0.02], seropositivity being found in 0.70 of sufferers. CONCLUSION: Familial transmission of chronic infection plus part of syndrome links Helicobacter with causality. Seropositivity not being universal throughout parkinsonism, consequent on gastric atrophy +/- sporadic antibiotic exposure, might explain less aggressive disease in older sufferers.

L20 ANSWER 27 OF 31 MEDLINE
AN 1998118370 MEDLINE
TI SIDS, licensed care centers, and Helicobacter pylori.
AU Pattison C P; Marshall B J
SO PEDIATRICS, (1998 Feb) 101 (2) 324.
Journal code: 0376422. ISSN: 1098-4275.

L20 ANSWER 28 OF 31 MEDLINE
AN 1998083577 MEDLINE

TI Proposed link between Helicobacter pylori and sudden infant death syndrome.
AU Pattison C P; Marshall B J
SO MEDICAL HYPOTHESES, (1997 Nov) 49 (5) 365-9. Ref: 52
Journal code: 7505668. ISSN: 0306-9877.
AB Helicobacter pylori may be linked to sudden infant death syndrome (SIDS) through synthesis of inflammatory cytokines, particularly interleukin-1, which can produce fever, activation of the immune system, and increased deep sleep. A relatively minor respiratory or enteric infection, together with overwrapping and prone sleep position could then induce terminal hypoxemia. Alternatively, H. pylori produces large amounts of urease which, if aspirated in gastric juice, could reach the alveolae, react with plasma urea, and produce ammonia toxicity leading to respiratory arrest. Epidemiological similarities between H. pylori and SIDS are presented along with possible transmission mechanisms for H. pylori which support this hypothesis.

L20 ANSWER 29 OF 31 MEDLINE
AN 97109527 MEDLINE
TI Gastric Helicobacter pylori infection as a cause of idiopathic Parkinson disease and non-arteric anterior optic ischemic neuropathy.
AU Altschuler E
SO MEDICAL HYPOTHESES, (1996 Nov) 47 (5) 413-4.
Journal code: 7505668. ISSN: 0306-9877.
AB The mechanisms of pathogenesis for both idiopathic Parkinson disease and non-arteritic anterior optic ischemic neuropathy are unknown. A study has shown that, in both diseases, there is a higher prevalence of gastrointestinal ulcers than in age- and sex-matched controls or than in the reported rates for the general population. It is proposed that gastric Helicobacter pylori infection may be a cause of both these diseases.

L20 ANSWER 30 OF 31 MEDLINE
AN 96173857 MEDLINE
TI A 35-year-old man with epigastric pain, 1 year later.
AU Delbanco T L; Daley J
SO JAMA, (1996 Mar 6) 275 (9) 722.
Journal code: 7501160. ISSN: 0098-7484.

L20 ANSWER 31 OF 31 MEDLINE
AN 95356323 MEDLINE
TI A 35-year-old man with epigastric pain.
AU Glickman R
SO JAMA, (1995 Aug 9) 274 (6) 495-500.
Journal code: 7501160. ISSN: 0098-7484.

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